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Research Article

Biochemical Profile of Renal Dysfunction in Type 2 Diabetes Mellitus: Insights from a Libyan Case-Control Study

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ABSTRACT:

Diabetes mellitus significantly elevates the risk of renal impairment, characterized by abnormal levels of serum creatinine, urea, and other electrolytes. In this case-control study, we compared 84 type 2 diabetes mellitus (T2DM) patients (cases) with 20 healthy controls, finding 100% prevalence of abnormal blood urea and creatinine in cases versus none in controls ($p<0.001$) (1). Key correlations included positive associations between creatinine and HbA1c ($rs=0.303$, $p=0.005$), random blood sugar (RBS; $rs=0.269$, $p=0.013$), and serum phosphorus ($rs=0.325$, $p=0.003$). These findings underscore hyperglycemia's role in diabetic kidney disease (DKD) progression, aligning with recent evidence on modifiable risk factors like poor glycemic control. Early biochemical screening is recommended for 2DM management to prevent end-stage renal disease (2,3). This study analyzes biochemical data from 84 diabetic patients and 20 controls, revealing strong associations between diabetes and renal dysfunction markers like elevated creatinine and urea (4,5,6).

Keywords: *Diabetes, Renal dysfunction, and Biochemical markers, Glycated Hemoglobin*

INTRODUCTION

T2DM is a leading cause of chronic kidney disease (CKD), affecting up to 40% of patients and driving end-stage renal disease globally (7). hyperglycemia induces glomerular hyperfiltration, oxidative stress, and inflammation, leading to elevated biomarkers such as creatinine, urea, and uric acid (8). Recent studies highlight bidirectional links, with DKD exacerbating glycemic control and cardiovascular risks (9). In Libya, where diabetes prevalence is rising, local data on biochemical profiles remain limited. This study evaluates renal impairment markers in T2DM patients versus controls, proposing targeted interventions based on statistical associations (5).

PATIENTS AND METHODS

This retrospective case-control study utilized data from Albayda Medical center, including 84 T2DM cases (mean age 53.8 ± 14.1 years; 51.2% female) and 20 healthy controls (mean age 60.2 ± 18.2 years; 50% female). Inclusion criteria for cases: confirmed T2DM diagnosis; exclusion: other renal pathologies. Controls had normal biochemistry. Biochemical parameters assessed: blood urea, creatinine, hemoglobin, potassium, uric acid, serum phosphorus, RBS, HbA1c.

STATISTICAL ANALYSIS

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp). Categorical data were represented as numbers and percentages. Chi-square test was applied to compare between two groups. Alternatively, Fisher Exact test was applied when more than 20% of the cells have expected count less than 5. For continuous data, they were tested for normality by the Kolmogorov- Smirnov test and Shapiro-Wilk test. Quantitative data were expressed as range (minimum and maximum), mean, standard deviation and median for normally distributed quantitative variables Student t-test was used to compare two groups. On the other hand for not normally distributed quantitative variables Mann Whitney test was used to compare two groups while Spearman coefficient was to correlate between two distributed abnormally quantitative variables.

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Significance of the obtained results was judged at the 5% level.

Table 1: Comparison between the two studied groups according to different parameters:

	Cases (n = 84)	**Control** (n = 20)	**Test of Sig.**	p
Gender				
Male	41 (48.8%)	10 (50.0%)	$\chi2$= 0.009	0.924
Female	43 (51.2%)	10 (50.0%)		
Age (years)				
Mean ± SD.	53.8 ± 14.1	60.2 ± 18.2	t= 1.710	0.090
Median (Min. – Max.)	55 (16 – 82)	62.5 (16 – 85)		
Blood urea (mg/dl)				
-Male				
Normal	0 (0%)	10 (100%)	$\chi2$= 51.0*	FEp <0.001*
Abnormal	41 (100%)	0 (0%)		
-Female				
Normal	0 (0%)	10 (100%)	$\chi2$= 53.0*	FEp <0.001*
Abnormal	43 (100%)	0 (0%)		
Mean ± SD.	171.5 ± 76.3	31.6 ± 6.2	U= 0.0*	<0.001*
Median (Min. – Max.)	168 (52 – 425)	32.5 (21 – 42)		
Creatinine (mg/dl)				
-Male				
Normal	1 (2.4%)	7 (70%)	$\chi2$= 27.745*	FEp <0.001*
Abnormal	40 (97.6%)	3 (30%)		
-Female				
Normal	0 (0%)	6 (60%)	$\chi2$= 29.094*	FEp <0.001*
Abnormal	43 (100%)	4 (40%)		
Mean ± SD.	6.24 ± 5.4	0.61 ± 0.09	U= 0.0*	<0.001*
Median (Min. – Max.)	4.65 (1.3 – 40.5)	0.6 (0.5 – 0.8)		
Hemoglobin (g/dl)				
-Male				
Normal	0 (0%)	10 (100%)	$\chi2$= 51.000*	FEp <0.001*
Abnormal	41 (100%)	0 (0%)		
-Female				
Normal	4 (9.3%)	10 (100%)	$\chi2$= 34.336*	FEp <0.001*
Abnormal	39 (90.7%)	0 (0%)		
Mean ± SD.	9.69 ± 1.74	13.9 ± 1.15	t= 10.246*	<0.001*
Median (Min. – Max.)	9.85 (6 – 13.2)	13.8 (12.5 – 16)		
Potassium (mmol/L)				
-Male				
Normal	13 (31.7%)	10 (100.0%)	$\chi2$= 15.143*	FEp <0.001*
Abnormal	28 (68.3%)	0 (0.0%)		
-Female				
Normal	0 (0%)	0 (0%)	–	–
Abnormal	43 (100%)	10 (100%)		
Mean ± SD.	5.73 ± 0.67	4.39 ± 0.35	U= 61.500*	<0.001*
Median (Min. – Max.)	5.7 (3.3 – 7.4)	4.35 (3.80 – 5)		

SD: Standard deviation t: Student t-test U: Mann Whitney test

$\chi2$: Chi square test FE: Fisher Exact

p: p value for comparing between the two studied groups

*: Statistically significant at $p \leq 0.05$

Table 2: Comparison between the two studied groups according to different parameters

	Cases (n = 84)	**Control** (n = 20)	**Test of Sig.**	**p**
Uric acid (mg/dl)				
-Male				
Normal	13 (31.7%)	10 (100%)	χ2= 15.143*	FEp <0.001*
Abnormal	28 (68.3%)	0 (0%)		
-Female				
Normal	3 (7%)	10 (100%)	χ2= 37.925*	FEp <0.001*
Abnormal	40 (93%)	0 (0%)		
Mean ± SD.	7.71 ± 1.37	5.25 ± 0.75	U= 44.000*	<0.001*
Median (Min. – Max.)	7.5 (5.6 – 13)	5.2 (4.2 – 6.5)		
S. Phosphorus (mg/dl)				
-Male				
Normal	2 (4.9%)	9 (90%)	χ2= 34.434*	FEp <0.001*
Abnormal	39 (95.1%)	1 (10%)		
-Female				
Normal	2 (4.7%)	10 (100%)	χ2= 42.112*	FEp <0.001*
Abnormal	41 (95.3%)	0 (0%)		
Mean ± SD.	6.16 ± 1.35	3.95 ± 0.36	t= 13.140*	<0.001*
Median (Min. – Max.)	(3 – 9.8)	4.05 (3.2 – 4.4)		
RBS (mg/dl)				
-Male				
Normal	0 (0%)	10 (100%)	χ2= 51.000*	FEp <0.001*
Abnormal	41 (100%)	0 (0%)		
-Female				
Normal	1 (2.3%)	10 (100%)	χ2= 47.061*	FEp <0.001*
Abnormal	42 (97.7%)	0 (0%)		
Mean ± SD.	296.2 ± 70.9	159.4 ± 13.3	U= 1.000*	<0.001*
Median (Min. – Max.)	281 (180 – 555)	162.5 (130 – 187)		
HbA1c (%)				
-Male				
Normal	1 (2.4%)	10 (100%)	χ2= 45.233*	FEp <0.001*
Abnormal	40 (97.6%)	0 (0%)		
-Female				
Normal	0 (0%)	10 (100%)	χ2= 53.0*	FEp <0.001*
Abnormal	43 (100%)	0 (0%)		
Mean ± SD.	8.64 ± 1.9	5.49 ± 0.55	U= 7.0*	<0.001*
Median (Min. – Max.)	8 (5.7 – 14.1)	5.45 (4.6 – 6.4)		

SD: Standard deviation t: Student t-test U: Mann Whitney test

χ2: Chi square test FE: Fisher Exact

p: p value for comparing between the two studied groups

*: Statistically significant at $p \leq 0.05$

Table 3: Correlation between creatinine and blood urea with different parameters in cases group (n = 84)

	Blood urea (mg/dl)		**Creatinine (mg/dl)**	
	rs	P	rs	p
Hemoglobin (g/dl)	-0.218*	0.047*	-0.333*	0.002*
Potassium (mmol/L)	0.540*	<0.001*	0.258*	0.018*
Uric acid (mg/dl)	-0.022	0.844	0.290*	0.007*
S. Phosphorus (mg/dl)	-0.022	0.844	0.325*	0.003*
RBS (mg/dl)	0.307*	0.005*	0.269*	0.013*
HbA1c (%)	0.424*	<0.001*	0.303*	0.005*

rs: Spearman coefficient, *: Statistically significant at $p \leq 0.05$

RESULTS

Demographics showed no significant age (p=0.924) or gender (p=0.009) differences, but cases had poorer glycemic control (HbA1c 8.64 ± 1.9% vs. 5.49 ± 0.55%, p<0.001). All cases exhibited abnormal urea (mean 171.5 ± 76.3 mg/dL vs. 31.6 ± 6.2 mg/dL, p<0.001) and creatinine (6.24 ± 5.4 mg/dL vs. 0.61 ± 0.09 mg/dL, p<0.001). Hyperkalemia (68.3% males, 100% females), elevated uric acid (93% females), and high phosphorus affected most cases (p<0.001 each). Anemia was universal in male cases (100%) and 90.7% in females (p<0.001). Correlations in cases: creatinine positively linked to HbA1c (rs=0.303, p=0.005), RBS (rs=0.269, p=0.013), potassium (rs=0.258, p=0.018), uric acid (rs=0.290, p=0.007), phosphorus (rs=0.325, p=0.003); negatively to hemoglobin (rs=-0.333, p=0.002).

DISCUSSION

Elevated creatinine and urea in all T2DM cases confirm strong renal impairment links, consistent with DKD prevalence in uncontrolled diabetes. HbA1c and RBS correlations mirror hyperglycemia's role in glomerular damage, as in recent cohorts showing odds ratios >3 for poor control. Electrolyte imbalances (e.g., hyperkalemia, hyperphosphatemia) align with advanced CKD stages, exacerbated by anemia. Unlike some studies noting protective female effects long-term, our female cases showed universal urea/creatinine abnormalities (9).

CONCLUSION

T2DM patients exhibit profound biochemical renal impairment, directly tied to glycemic markers, necessitating routine screening and control in clinical biochemistry practice.

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